U.S. Department of Labor

Office of Administrative Law Judges
Seven Parkway Center - Room 290
Pittsburgh, PA 15220

(412) 644-5754
(412) 644-5005 (FAX)

Issue Date: 23 August 2005

Case No. 2004-BLA-5617

In the Matter of:

ELEANORA M. VOYTEN,
Survivor of JOHN ANDREW VOYTEN,
Claimant,

v.

CONSOLIDATION COAL COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances:

Robert J. Bilonick, Esq.
For the Claimant

William S. Mattingly, Esq.
For the Employer

Before: MICHAEL P. LESNIAK
Administrative Law Judge

DECISION AND ORDER — AWARDING BENEFITS

This proceeding arises from a survivor's claim filed by Eleanora M. Voyten, the surviving spouse of John Voyten ("Miner"), for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq.* (the Act) and its implementing regulations set forth in Title 20 of the Code of Federal Regulations. Benefits under the Act are awarded to persons who are totally disabled due to pneumoconiosis and to the survivors of persons whose death was caused by coal workers' pneumoconiosis (CWP). Pneumoconiosis is a chronic dust disease of the lungs, including respiratory and pulmonary impairments arising out of coal mine employment, and is commonly referred to as black lung.

In accordance with the Act, this case was referred by the District Director to the Office of Administrative Law Judges. A formal hearing was conducted in Pittsburgh, Pennsylvania, on December 8, 2004, at which time all parties were given a full opportunity to present evidence and argument as provided in the Act and applicable Regulations. The record consists of Director's Exhibits 1–36, Claimant's Exhibits 1–4, and Employer's Exhibits 1–11, as well as testimony from the Claimant and the Miner's son.[1]

The findings of fact and conclusions of law that follow are based upon my analysis of the administrative record, including all documentary evidence admitted, testimony at the formal hearing, and the arguments of counsel.

PROCEDURAL HISTORY

The Miner filed two claims for benefits under the Act. The first claim was filed on March 18, 1983, and was finally denied on July 8, 1983 because Mr. Voyten failed to establish total disability due to pneumoconiosis. (DX 1). Mr. Voyten filed a second living miner's claim on October 16, 1985. This claim was finally denied on March 18, 1986 for the same reasons. (DX 2). No further action was taken on either claim.

The Miner died on July 13, 2002 at the age of eighty-one. (DX 4, 12). On December 6, 2002, Claimant filed the instant survivor's claim. (DX 4). On April 18, 2003, the District Director issued a *Schedule for the Submission of Additional Evidence*. The District Director indicated that, given the state of the record at that time, Claimant would be entitled to benefits. (DX 25). The District Director on September 30, 2003 issued a *Proposed Decision and Order – Award of Benefits – Responsible Operator.* (DX 27). The Employer requested a formal hearing by letter dated October 31, 2003. (DX 30). On January 6, 2004, this claim was referred to this Office for a formal hearing. (DX 34). The hearing was conducted as noted above before the undersigned on December 8, 2004 at Pittsburgh, Pennsylvania.

ISSUES

The sole matter at issue in this case is whether the Miner's death was due to pneumoconiosis.

At the formal hearing, the Employer withdrew its opposition to its designation as responsible operator. (Tr. 17). There is no dispute as to whether there exist additional dependents for the augmentation of benefits.

---

[1] The following references are used herein: TR for transcript, CX for Claimant's Exhibit, DX for Director's Exhibit, and EX for Employer's exhibit. With respect to the exhibits from the living miner's claims, I find good cause for their admission. Although the record in a miner's claim is not always admissible, I find that their admission would assist in the evaluation of the medical opinion evidence in this case. There was no objection to the admission of the Employer's exhibits. (Tr. 5). To the extent the exhibits introduced by the Employer exceed the evidentiary limitations set for in the Secretary's regulations, I find that good cause exists for their admission as well.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Applicable Law

Because the Miner was last employed in the coal industry in the Commonwealth of Pennsylvania, this claim is governed by the law of the United States Court of Appeals for the Third Circuit. *See Shupe v. Director*, *OWCP*, 12 B.L.R. 1-200 (1989) (*en banc*).

Coal Mine Employment

Claimant has asserted over thirty years of coal mine employment. (DX 4; *see* Tr. 7). The Employer has also stipulated to at least fifteen years of coal mine employment. (Tr. 8). Based on Claimant's testimony, in which she recounted that Mr. Voyten had worked during the early years of their marriage, I credit the Miner with at least twenty years of qualifying coal mine employment.

Hearing Testimony

Both the Claimant and the Miner's son, Mr. John A. Voyten, testified at the hearing. Mrs. Voyten testified that she and Mr. Voyten were married in 1942. Her husband was working in the mines at that time, and had been so occupied since 1939. (Tr. at 10–11). She also recalled that her husband was a smoker, having smoked cigarettes at the rate of one-half pack per day for thirty-five years. (Tr. at 11). Mr. Voyten stopped working in the mines in 1982 or 1983 when the mine shut down. He had experienced some difficulty breathing during the 1970s, and his breathing "varied" after he left the mines. (Tr. at 12). His breathing curtailed some activities. According to Mrs. Voyten, her husband liked to garden and had difficulty doing this work because he could not bend over. (Tr. at 12).

The Miner's son, John Voyten, who is also a coal miner, testified that his father last worked at the Renton mine in 1982. (Tr. at 14). He emphasized that, because the Miner had suffered a heart attack at age sixty-two just before leaving the mines, his father stopped smoking at that time. (Tr. at 18). The son also affirmed that his father's breathing was not that good, and recalled that the elder Mr. Voyten had breathing difficulties exacerbated by humidity and underground work. (*Id*.).

Medical Evidence

Death Certificate

The Miner's death certificate was certified by Dr. S. Bajwa on July 15, 2002. (DX 12). Dr. Bajwa certified the immediate cause of death as "metastatic carcinoma of colon," with pneumoconiosis as an "[o]ther significant condition[] contributing to death."

Autopsy

An autopsy, with the examination restricted to the Miner's chest, was conducted on July 14, 2002 by Dr. Curtis Goldblatt, who was assisted by Dr. Chakraverty. (DX 13). The "Final Anatomic Diagnoses" were listed as:

1. Right Lower Lobe Bronchioalveolar Carcinoma, Mucinous Type (2.5 cm)
2. Mixed Macro and Micronodular Coal Workers' Ppneumoconiosis
3. Bullous Emphysema
4. Cor Pulmonale (Right Ventricle 1.3 cm)
5. Pulmonary Congestion and Edema (Right 1,050 Grams, Left 890 Grams)
6. Cardiomegaly (530 Grams)
7. Old Myocardial Infarct Scars
8. Severe Atherosclerotic Coronary Artery Disease

The prosectors rendered the following "Clinicopathological Summary":

> Autopsy ... reveals a right lower lobe bronchioloalvealoar carcinoma, mucinous type. Simple coal worker's pneumoconiosis, severe pulmonary emphysema, cor pulmonale and artheriosclerotic coronary artery disease with old myocardial infarcts contributed to his demise.

Dr. Goldblatt detected on gross examination of the chest that the hilar lymph nodes showed, *inter alia*, anthracosis and "metastatic involvement." The pleural surfaces of the lungs included black macules measuring up to 0.4 cm in diameter. These covered about fifteen percent of the lung surfaces. The prosector also discovered in sections of the pulmonary parenchyma "numerous black nodules" among other findings, with the largest measuring 0.7 cm.

Dr. Goldblatt also conducted a microscopic examination, and in slides from the lungs found:

> There is a 2.5 cm, mucinous-type, bronchioloalveolar carcinoma in the right lower lobe .... There are diffuse intra-alveolar edema and congestion. There are thickened hyalinized vessels of small and medium-sized arteries. There is alveolar wall space enlargement with septal spurring and destruction of the alveolar walls in a centrilobular pattern consistent with emphysema. The interstitium exhibits fibrosis, nodules up to 0.7 cm and anthracotic pigment. Collections of anthrasilicotic pigment laden macrophages (macules) measuring 0.2 cm to 0.4 cm in diameter are seen in the subpleural, paraseptal, perivascular and peribronchial regions involving 15 percent of the parenchyma, predominately in the upper lobes. Polarized crystals morphologically similar to silica are present within the macules.

Dr. Goldblatt is board certified in anatomical and clinical pathology. (DX 13). Dr. Chakraverty was a pathology resident at the time of this post-mortem examination. (*Id*.).

Dr. Goldblatt provided deposition testimony on November 16, 2004. (EX 11). At the outset, he indicated that additional information had come to his attention in the form of results of two tests, or markers B CX20 and CX7 B, that would change his opinion to some extent. (EX 11 at 21). These markers may suggest the source of the cancer that was found in the Miner's lung. The markers indicated to Dr. Goldblatt that the Miner had a "gastrointestinal primary," consistent with metastases to the lung. (EX 11 at 21). Given this source, the doctor opined that there would be no association between coal mine dust exposure and the lung cancer. (EX 11 at 23).

Although there was simple pneumoconiosis, there was no evidence of progressive massive fibrosis or lesions. Dr. Goldblatt was asked about the extent of the Miner's impairment. He had reviewed the results of clinical studies and noted that Dr. Klemens had found Mr. Voyten to be totally disabled. He acknowledged, however, that Dr. Klemens's assessment was without objective support save for a positive x-ray. (*Id*. at 33).

The Miner's heart was "biventricularly enlarged," with areas of prior myocardial infarction. (EX 11 at 39). The scars that resulted from the MI were deemed to be "enormous." (*Id*. at 40). Nevertheless, although the heart was biventricularly enlarged, the doctor emphasized that the Miner still had cor pulmonale. Dr. Goldblatt explained that "the right ventricle was much more chronically thickened than the left ventricle. In addition, he also had changes in [the] small and medium sized blood vessels in his lung that would indicate increased vascularzation." (*Id.* at 44). Dr. Goldblatt agreed that cor pulmonale would generally be accompanied by abnormalities in arterial blood transfer. Yet there were no recent tests to show hypoxemia (*Id*. at 45). Because there were no recent arterial blood gas test results, Dr. Goldblatt did not consider the fact that the tests did not document "profound hypoxemia" to be "determinative." (*Id*. at 45)

In addition to the black macules, the doctor also found nodules that measured up to seven millimeters. About fifteen percent of the lung surface was covered by macules of this nature. (*Id.* at 46–47). He opined that the macular changes were associated with nodules, which suggest pulmonary effects or changes beyond those associated with the macule alone. (*Id.* at 48). He testified that macules made up a greater percentage, "15 percent," but "in terms of being palpable, that would be the nodules." (*Id.* at 47).

With respect to the Miner's emphysema, Dr. Goldblatt thought that thirty years in the mines and smoking would combine to produce pulmonary emphysema, which in this case was centrilobular and bullous emphysema. (*Id.* at 49–50). He could exclude neither coal mine dust exposure nor smoking as causes of the emphysema. In any event, the Miner suffered from a severe degree of emphysema. (*Id.* at 55). Dr. Goldblatt thought that bullous emphysema could be a manifestation of centrilobular emphysema. The emphysema that afflicted Mr. Voyten was severe. The physician cited testimony to the effect that the Miner had suffered from shortness of breath. (*Id.* at 55–56).

Dr. Goldblatt was asked whether an autopsy prosector might have an advantage over a reviewing physician with respect to the interpretation of pathology slides. He responded that the reviewer might have a "slight disadvantage because he wasn't there when the individual samples

were taken. So he didn't see them grossly, the gross microscopic correlation, direct microscopic correlation for each slide."[2] (*Id.* at 57–58).

When asked to provide a the cause of death, Dr. Goldblatt noted that only two organs were presented for review, although other organs could have been involved in the Miner's death. (*Id.* at 60). He nevertheless opined:

> The lung disease, including the coal workers' pneumoconiosis, the severe bullous pulmonary emphysema and the heart disease that arose from the lung disease, the cor pulmonale, would all contribute to producing myocardial ischemia. In a person who already had an abnormal heart which has previously had severe scarring and damage, he would be very susceptible to developing cardiac arrhythmia due to lack of sufficient oxygen to the heart.

(*Id.* at 61). Thus, the Miner's pulmonary condition may have precipitated an arrhythmia, in Dr. Goldblatt's opinion. Because of the limited autopsy, he could not provide an opinion as to the contribution of cancer to the Miner's death. (*Id.* at 62). Dr. Goldblatt thought that the clinical history and Dr. Perper's opinion supported his conclusions. (*Id.* at 65). He summarized that the slight abnormalities observed in the Miner's pulmonary function yen years prior to death

> indicates the beginning of a significant lung disease. And the significant lung disease primarily from his occupational exposure led to hypoxemia, which led to decreased oxygen supply to the heart when the heart was already impaired significantly due to previous injury. That impaired oxygen to the heart produced abnormal heart function, specifically abnormal cardiac rhythms, which ultimately led to cessation of heart function.

(*Id.* at 70–71). Dr. Goldblatt voiced his agreement with the opinions of Dr. Bajwa and Dr. Perper. (*Id.* at 64–65).

Medical and Treatment Records – Records from Prior Claims

Claimant has submitted voluminous medical records from the Miner's various hospitalizations and treatment for a variety of illnesses throughout the years. Many of these records were generated during the period when Mr. Voyten had filed his living miner's claims for benefits under the Act. I have admitted the records from the earlier claims into evidence, as well as the treatment records. The treatment records place in context discussions about the extent of any pulmonary or respiratory difficulties experienced by the Miner. These records, and evidence generated for the miner's claims, also form some of the documentation for the current medical opinions. Although these records will not be set forth separately, I have reviewed them and have considered them in rendering this decision.

[2] Dr. Tomashefski took a different view, testifying that the prosector would enjoy no advantage in reviewing the slides. (EX 8 at 14).

Dr. Joshua A. Perper

Dr. Perper conducted a comprehensive evaluation based on his review of thirty slides, the autopsy protocol and other medical records. He presented his conclusions in a report dated May 17, 2003. Dr. Perper outlined his microscopic findings, which included:

1. The pleura shows focal slight to moderate fibro-anthracosis, with presence of birefringent silica crystals.
2. Scattered throughout the pulmonary parenchyma are scattered anthracotic macules located around blood vessels, airways and in the inter-alveolar septa. Birefringent silica crystals are seen within the macules. ...
3. Scattered throughout the pulmonary parenchyma are scattered mixed coal dust micronodules and ... a few silicotic type micronodules, with an occasional macronodule. The micronodules measure up to 4–5 mm in maximal dimension and show peripheral focal (scar) emphysema. ...
4. Severe centrilobular emphysema and interstitial fibrosis.
5. Two of the lung sections are largely replaced by adenocarcinoma glandular structures with copious mucin production. ...
6. Slight sclerosis of intra-pulmonary blood vessels, consistent with pulmonary hypertension.
7. One lung section had a focus of metaplastic calcification/ossification.
8. Pulmonary lymph nodes: fibro-anthracosis, silicotic nodules and foci of metastatic adeoncarcinoma.

Dr. Perper rendered the following microscopic diagnoses:

1. Coal workers' pneumoconiosis, simple, micronodular, slight to moderate severity.
2. Centrilobular emphysema, severe, with interstitial fibrosis.
3. Mucinous adenocarcinoma, right lower lobe.
4. Sclerosis of small intra-pulmonary blood vessels consistent with pulmonary hypertension.
5. Myocardial infarction scarring, remote.

Dr. Perper's report also incorporates microphotographs that illustrate his findings. He then concluded that the Miner suffered from pneumoconiosis; he opined with respect to the role of this disease in the Miner's death:

> Based on the autopsy findings indicative of significant coal workers pneumoconiosis of slight to moderate severity, his long-standing occupational history of coal mining and exposure to coal mine dust as discussed above, it is my professional opinion ... that coal workers' pneumoconiosis was a significant contributory cause of death of Mr. Voyten (along with his other causes of death) and a hastening factor of his death, both directly and indirectly through direct replacement of normal lung tissue by pneumoconiotic lesions and associated

centrilobular chronic emphysema and resting hypoxemia, which was also demonstrated clinically.

The mechanism of death contributed by the presence of coal workers' pneumoconiosis was through the following pathways:

1. Direct pulmonary insufficiency due to replacement of normally breathing lung by non-breathing pneumoconiotic tissues and associated centrilobular emphysema, and resulting hypoxemia.
2. [H]ypoxemia precipitating/aggravating a cardiac arrhythmia in an individual with heart disease.

Thus, Dr. Perper was able to conclude in summary that:

> Coal workers' pneumoconiosis, and the associated centrilobular emphysema, was a substantial contributory cause of Mr. Voyten's death both directly and indirectly through pulmonary insufficiency and through hypoxemia triggering or aggravating an arrhythmia, on the background of marked heart disease.

Dr. Perper's deposition was recorded on May 10, 2004. (CX 1). He reiterated his microscopic diagnosis of simple coal worker's pneumoconiosis, micronodular, of "mostly slight to moderate severity." Mr. Voyten also suffered from centrilobular emphysema and interstitial fibrosis. Dr. Pickerill found no evidence of acute histoplasmosis or resultant pneumonitis. The Miner suffered from cancer. Dr. Perper identified a "mucinous carcinoma," but could not verify whether the cancer was a primary tumor or was secondary to the Miner's colon cancer. (*Id.* at 10). He further opined that the multiple metastases that were identified on chest x-ray films were "nodulars of coal workers' pneumoconiosis." (*Id.* at 12). He thought that none of the reviewing pathologists identified any metastatic cancerous nodules other than the primary mass. (*Id*.). Dr. Perper also explained his finding of cor pulmonale. (*Id.* at 17).

On cross-examination, Dr. Perper explained his opinion that the mechanism of death was direct pulmonary insufficiency:

> [T]here is definitely a decrease in the capability of exchange of gasses[.] ... [T]here was also most fairly constant finding of low-to-low level of normal of exchange of oxygen which decreased even more with exercise. ... [F]or a number of years there ... were no pulmonary function tests taken and/or arterial blood gases, but on the other hand the severity of the coal workers' pneumoconiosis and of the emphysema at the autopsy in my opinion substantiates my statement.
>
> * * *
>
> [T]he chest x-ray shows evidence of severe chronic obstructive pulmonary disease, which is consistent with hypoxemia and ... there is a trend which is pretty consistent that his lower normal level became even lower with exercise.

* * *

> [T]here is evidence of severe chronic obstructive pulmonary disease both clinically and documented at autopsy. There is testing to show that exercise decreases the patient's pO2 and in addition to the chronic shortness of breath, which is evident in almost every one of his examinations. So, based on those and based on the pathology of the lungs, that is my conclusion.

(*Id.* at 24–26).

He explained his conclusion that the Miner suffered from chronic obstructive pulmonary disease by pointing to the findings on autopsy of centrilobular emphysema. He attributed the Miner's shortness of breath to his lung disease, because that symptom had first been documented in 1983, before Mr. Voyten had been in congestive heart failure, and other causes of shortness of breath "were not documented in this individual." (*Id.* at 34). Dr. Perper found no clinical symptoms of arteriosclerotic heart disease at the time the Miner first complained of shortness of breath before 1985. (*Id.* at 37).

Dr. Perper explained that the mineral dust he saw was silica because it contained "very tiny birefringent crystals." (*Id.* at 48). With respect to the Miner's emphysema, the doctor concluded that one could not differentiate between emphysema due to coal mine dust exposure and smoking. The pneumoconiosis was also progressive in this case

> [b]ecause the statements of the miner himself that he has shortness of breath worsened over the year. Second, ... the radiological findings certainly became much worse and [third] he stopped smoking about 50 years before his death, but when he was diagnosed radiographically they found severe chronic obstructive lung disease ... and finally the autopsy findings.

(*Id.* at 49–50). With respect to the cor pulmonale, Dr. Perper noted that the hypertrophy of the right ventricle was much larger than that of the left. He distinguished the anthracotic pigmentation found in the public at large from that found with pneumoconiosis: "[P]eople who are living in the city have a silicotic pigmentation, but they don't have anthracotic pigmentation to the same extent and they don't have anthracotic pigmentation in conjunction with silica crystal deposition." (*Id.* at 53).

On re-direct examination, Dr. Perper elaborated on his opinion regarding the distinction between the emphysema due to smoking and that derived from pneumoconiosis:

> [T]here are two processes which cause centrilobular emphysema. One is exposure to smoke and the other is coal workers' pneumoconiosis. He was a significant smoker, however he stopped 50 years before his death and usually there is no ... progression in centrilobular emphysema in smokers once they stop smoking.

> On the other hand, he also had centrilobular emphysema which is due to coal workers' pneumoconiosis and this continues after cessation of exposure from coal mine dust.
>
> ... [T]hey look identical. Only their causative and etiological mechanism is different.

(*Id.* at 55–56). Dr. Perper also opined that cor pulmonale and hypertensive disease can coexist. If the right-sided hypertrophy were secondary to the left ventricular hypertrophy, then both sides of the heart would be similar in size. In this case, the right sided enlargement is greater and would be due to the Miner's lung disease. (*Id.* at 58).

Finally, Dr. Perper was asked about the Miner's medications, which included inhalers. He stated that there is no treatment for pneumoconiosis, but that such medications can afford symptomatic relief.

Dr. Perper is the chief medical examiner for Broward County in Fort Lauderdale, Florida, and is board-certified in forensic pathology and anatomical and surgical pathology. Dr. Perper holds a current professorship in pathology at the University of Miami and was a clinical professor of pathology at the University of Pittsburgh. (*Id.* at 4–5).

Dr. Surinder S. Bajwa

Dr. Bajwa had been treating the Miner for a number of years. He submitted a brief letter report in which he opined, based on his review of the medical records, that "coal worker's pneumoconiosis substantially contributed to the death of Mr. John Voyten." (CX 3). Dr. Bajwa is board certified in internal medicine and pulmonary disease. (CX 4).

Dr. Everett F. Oesterling

Dr. Oesterling evaluated the record at the request of the Employer and submitted his consultation report on May 6, 2003. (DX 15). Dr. Oesterling reported that he had reviewed twenty-five histologic slides. Seventeen of the slides contained cross-sections of lung tissue, and Dr. Oesterling employed photomicrographs to illustrate his detailed findings and conclusions. The doctor concluded:

1. This gentleman did have coalworkers' pneumoconiosis, a very mild form of the disease process.
2. The change related to dust deposition is insufficient to have altered pulmonary function, thus it would have produced no respiratory impairment or disability during the miner's lifetime.
3. The cause of this gentleman's death will be illustrated in subsequent photos.
4. His death was not caused by, contributed to or hastened by any chronic dust disease arising out of his coalmine employment.

(DX 15). He described the effects of the Miner's smoking, in particular the cancerous tumor cells and the extensive mucous that resulted.

Dr. Oesterling disagreed with the primary diagnosis reached by the autopsy prosectors, who concluded that the Miner suffered from "Right Lower Lobe Bronchoalvolar Carcinoma, Mucinous type." Instead, Dr. Oesterling made the following primary diagnoses based on his findings in the lung tissue:

1. Metastatic colonic carcinoma.
2. Acute histoplasmosis with resultant pneumonitis complicating the metastatic tumor and resultant therapy.
3. Areas of fairly extensive pulmonary infarction.
4. Mild centrilobular pulmonary emphysema.
5. Minimal macular with focal micronodular coalworkers' pneumoconiosis.

The physician concluded that Mr. Voyten's death was due to "his metastatic disease and complications thereof and unrelated to his exposure to mine dust. Coalworkers' pneumoconiosis cannot be considered a factor in causing, hastening or contributing to his death." (DX 15).

In a letter, dated January 6, 2004, Dr. Oesterling reported on his review of additional slides. (EX 4). He thought that the additional slides showed "limited evidence of dust deposition," and classified the pneumoconiosis as a "mild macular coalworkers' pneumoconiosis." To Dr. Oesterling, the additional slides showed less evidence of mine dust exposure than those previously examined. He concluded, *inter alia*, that the level of pneumoconiosis "is insufficient to have altered pulmonary function, thus it produced no lifetime symptomatology." The "minimal change present due to mine dust exposure ... therefore did not contribute to death," Dr. Oesterling added.

Dr. Oesterling is board certified in clinical and anatomic pathology and nuclear medicine. (DX 15, EX 10 at 4).

Dr. Oesterling's deposition was taken on December 1, 2004. (EX 10). He was questioned about his previous reports and about five additional slides that were presented for his review. He noted that, with respect to these additional five slides, he saw "less coal workers' pneumoconiosis" than in the first group of slides. (*Id.* at 10).

Dr. Oesterling stated that the highest level of the pneumoconiosis that he saw was in the form of a macular lesion. (*Id.* at 15). He explained in detail a number of microphotographs that he had taken, and compared them to some obtained by Dr. Perper. Dr. Oesterling acknowledged that there were indications of pneumoconiosis, but concluded that there was "not a heavy amount" of "coal dust or coal pigment" in the lung tissues. (*Id*. at 21). He testified:

> If you [at] the first four photos, you can see the periphery of that nodule has some black pigment. When we put the polarized light into the field, these are very few crystals that we see in the coal dust. This is an old nodule. This is not an active area of fibrosis at this stage of the game.

> By contrast, the next four series of photographs shows a very definite macular area surrounding vessels. And when we go to the polarized light, there we see a greater abundance of silicate crystals, primarily; a few silica crystals as well.

(*Id.* at 21–22).

Dr. Oesterling opined that the cancer found in the lung resulted from a metastasis of the primary cancer that had afflicted the Miner's colon. The changes in the lungs that were due to pneumoconiosis were "insignificant." He explained:

> If we talk about the spectrum of coal workers' pneumoconiosis, macular change is the lowest form of the disease process that's truly a disease. When we have macular disease, normally it's insignificant. As I said, very heavy dust deposit possibly may produce some symptomatology; but normally, macules are, in and of themselves, not sufficient to alter function.

(Id. at 24). Dr. Oesterling also noted the diagnosis of bullous emphysema in the autopsy, a finding he considered to be consistent with spaces seen in the slides. He observed that bullous emphysema is a very severe form of emphysema. Coal mine dust exposure or pneumoconiosis do not cause bullous emphysema, in Dr. Oesterling's opinion. That form of emphysema is seen primarily in cigarette smokers or patients with congenital emphysema. (*Id.* at 27–28).

With respect to centrilobular emphysema, the doctor testified that it can be produced by coal mine dust exposure in the higher stages of pneumoconiosis. (*Id*. at 28). He opined that Mr. Voyten's exposure to coal mine dust was not responsible for the development of his centrilobular emphysema, explaining that he saw "very little or no coal mine dust in the areas" where he saw emphysema. (*Id*. at 29).

Dr. Oesterling also concluded that the Miner had significant heart disease. (*Id.* at 31). He disagreed with the prosector's finding of cor pulmonale, stating that the Miner's heart showed biventricular hypertrophy. Dr. Oesterling also noted that severe emphysema could also cause changes in the right ventricle. (*Id.* at 32, 34).

The doctor acknowledged that pneumoconiosis can progress after the cessation of exposure, but he opined that, in this case, there was no progression. (*Id*. at 37, 40). On cross-examination, he stated that only the higher stages of pneumoconiosis so progress. (*Id.* at 41, 42).

Dr. Oesterling opined that the cause of death would have been the metastatic carcinoma and its secondary effects. The physician clarified that Mr. Voyten did have "mild focal micronodular with macular" CWP. (*Id.* at 39). The pneumoconiosis "had nothing to do with the carcinoma that ultimately killed [the Miner]." (*Id*.). Dr. Oesterling also opined that the Miner's smoking likewise caused no disability, although bullous emphysema would have caused some symptoms. (*Id*. at 43). He also opined that Mr. Voyten was "developing a pneumonia due to histoplasmosis at the time of his death." The histoplasmosis in this case could have been an opportunistic infection as one complication of the cancer. (*Id.* at 46). The physician

acknowledged that a compromised pulmonary system would hasten death. (*Id*. at 47). He also conceded that the Miner had a "mildly compromised system."

Dr. Ben V. Branscomb

Dr. Branscomb reviewed a number of medical records at the request of the Employer; his report of this consultation was submitted on February 3, 2004. (EX 1). He referred to medical reports that had recorded at least twenty-nine years of coal mine employment and a smoking history of thirty or more years at the rate of three-quarters of a pack per day. To Dr. Branscomb, this was a "severe smoking history."

Dr. Branscomb, in commenting on the autopsy findings, called the prosector's finding of bronchioalveolar carcinoma a "reasonable mistake since [the prosector] did not know Mr. Voyten had proved metastatic colon cancer."

Dr. Branscomb took issue with Dr. Perper's interpretations and conclusions. He disagreed, for example, with Dr. Perper's statement that there were "respiratory problems requiring treatment with bronchodilators." According to Dr. Branscomb, there were "no valid studies showing the presence of any airways obstruction." He likewise did not accept Dr. Perper's view that all of the nodules in the lung were pneumoconiotic. In addition, Dr. Branscomb did not agree with Dr. Perper's attribution of microscopic changes to coal dust exposure rather than cigarette smoking. He explained that "lung volumes and spirometry up to the terminal illnesses demonstrated there was no functionally significant emphysema. Microscopic emphysema and also blebs are usual in old persons who have no pulmonary symptoms." Finally, Dr. Branscomb did not find any documentation of a pulmonary insufficiency such as would have hastened the Miner's death.

Dr. Branscomb concluded:

> I concur in the medical opinion that simple CWP is sometimes disabling, that CWP can be a progressive disorder first manifest after mining stops, that its manifestations may be latent, and that sometimes coal mine dust or CWP produce obstructive manifestations. I also incorporate in my definition of CWP for this report the concept that any pulmonary disorder or impairment in any way caused or significantly aggravated by either coal mine dust or CWP is regarded as pneumoconiosis. Further, I accept the concept that disability caused by non-occupational disorder which has been materially worsened by either coal mine dust or CWP is included as a disability attributable, at least in part, to CWP.
>
> * * *
>
> The pulmonary function studies and blood gas values reveal entirely normal function. This is consistent with the fact that in the hundreds of pages dealing with the terminal illnesses there was never any reference to significant pulmonary disease interfering in any way with the management and treatment. ... The finding of microscopic changes of emphysema in the lung is typical for an eighty-one

> year old long time smoker. However, there is no indication that either this or CWP produced any pulmonary impairment whatsoever.
>
> The description of the microscopic changes, the timing of the x-ray changes, and clinical circumstances generally are absolutely characteristic of death as a result of the cancer and its related complications. The severe coronary artery disease and previous infarction could have been a contributing factor although there is actually little information about the terminal events. The pulmonary congestion may be in part caused by the heart disease. There was manifestly insufficient scarring in the lungs judged by the pathology, x-ray changes, and function studies to produce cor pulmonale. For pulmonary hypertension to occur as the result of CWP extensive scars must block the blood flow through the lung. This was not present.
>
> I conclude that Mr. Voyten did have minimal simple CWP resulting from his coal mine employment. This caused no respiratory impairment or disability during his lifetime. Neither did it in any way accelerate his death or contribute to it. His death was caused by the inexorable progression of the highly invasive metastatic carcinoma of the lower colon. The usual complications of that disease and of its treatment were present. His death was neither caused by, hasten by, or contributed to by either any chronic disease arising out of coal mine employment or any chronic lung disease arising out of smoking or any other etiology.

(EX 1). Dr. Branscomb is board certified in internal medicine and has a lengthy academic career, including a professorship in respiratory diseases, and most recently has been a Distinguished Professor Emeritus of the University of Alabama at Birmingham. (EX 2).

Dr. Joseph F. Tomashefski, Jr.

Dr. Tomashefski prepared a consultative report on October 14, 2003, based on his review of the medical records. The Employer has submitted this report in rebuttal to the report prepared by Dr. Perper. (EX 3). Dr. Tomashefski concluded that

> Based on my review of the medical records and the slides prepared from Mr. Voyten's autopsy, it is my opinion that he had metastatic colonic adenocarcinoma, which had spread to his lung and hilar lymph nodes. ... I therefore disagree with the diagnosis of bronchioloalveolar carcinoma rendered in the autopsy report.
>
> It is also my opinion that Mr. Voyten had mild to moderate, predominately centriacinar, emphysema. In addition, there is a low-grade chronic interstitial pneumonitis of uncertain etiology.
>
> Based on the presence of a few pigmented micronodules, it is also my opinion ... that Mr. Voyten had minimal simple pneumoconiosis. The extent of this pneumoconiosis is so minimal, however, that it essentially represents an

> incidental finding of no clinical impact. Within reasonable medical certainty, Mr. Voyten's minimal simple coalworkers' pneumoconiosis neither caused nor contributed to his death. The minimal degree of anatomic coalworkers' pneumoconiosis is also supported by the clinical and radiological records.

(EX 3). Dr. Tomashefski also concluded that the Miner did not have pulmonary silicosis and that his pneumoconiosis did not cause his cancer, cardiovascular disease, aortic aneurism or emphysema. The doctor added that "[w]ithin reasonable medical certainty, Mr. Voyten's mild to moderate centrilobular emphysema was caused by his exposure to cigarette smoke for thirty years." He emphasized that the Miner's death was unrelated to coal mine employment. "Within reasonable medical certainty, [Mr. Voyten] would have died at the same time and in the same manner even if he had not worked in a coal mine or developed minimal simple coalworkers' pneumoconiosis."

Dr. Tomashefski disagreed with Dr. Perper's assessment of the extent of pneumoconiosis in this case, explaining that Dr. Perper is "overinterpreting perivascular deposition of pigment…[and that] the photomicrographs in Dr. Perper's report misrepresent the extent of simple coalworkers' pneumoconiosis." Dr. Tomashefski took issue with Dr. Perper's assessment of the extent of emphysema in this case, concluding instead that the Miner suffered from mild to moderate, rather than severe, emphysema. He also disagreed with Dr. Perper's finding of pulmonary hypertension, and lastly did not agree with the conclusion that the Miner's pneumoconiosis and coal mine dust exposure were causally related to the centriacinar emphysema. Dr. Tomashefski denied that the Miner's "minimal simple coalworkers' pneumoconiosis or mild to moderate emphysema is a cause or a contributory factor in his death." (EX 3).

He reiterated his conclusion in a letter report, dated July 2, 2004, after reviewing additional slides, that "Mr. Voyten would have died at the same time and in the same manner even if he had never worked as a coal miner or developed simple coalworkers' pneumoconiosis." (EX 5).

Dr. Tomashefski testified in a deposition recorded on November 18, 2004. (EX 8). He explained his disagreement with the prosector's finding of cor pulmonale:

> [T]he measurement of 1.3 centimeters is an exceedingly thick right ventricle of the type one sees in only the most severe degrees of pulmonary hypertension such as those caused by large left to right cardiac shunts in congenital heart disease. ... I'm skeptical of that measurement because it doesn't correlate with anything I see histologically or clinically that goes along with that severe degree of pulmonary hypertension. This man did not have the clinical features of cor pulmonale. He did not have venous neck vein engorgement, he did not have pitting edema of lower extremities and importantly in my review of the slides of his lungs he did not have disease anywhere severe enough to have caused that degree of right ventricular hypotrophy. His pulmonary arteries were in keeping with his age and the extent of parenchymal lung disease was very mild. Certainly nothing that would have caused that degree of right ventricular hypotrophy.

(EX 8 at 27–28).

The doctor pronounced as "trivial" the significance of the macular and micronodular disease he attributed to coal mine dust exposure, because "the lesions were very small ... [and] were very widely scattered over the lung such they comprised only about 1% ... of all the tissue I reviewed on the slides." (*Id.* at 29–30). With respect to the emphysema, Dr. Tomashefski considered it to be mild to moderate in degree because in his estimate it invovled only ten percent of the lung tissue. That degree, in his opinion, "minimally or not at all" affected the Miner's lung function. (*Id.* at 31).

Dr. Tomashefski concluded that the emphysema that was present was derived from the Miner's smoking. He explained that "there was no special relationship between the emphysema tissue lesions and the coal macules and micronodules .... Nor was there any particular special relationship between the deposits of coal dust and the emphysema lesions." (*Id.* at 31). He thus concluded that the emphysematous lesions were not caused by deposits of coal dust but were rather attributable to smoking. Nor did coal dust cause the Miner's pneumonitis. (*Id.* at 32).

The doctor opined that the Miner's death was sudden, but said that he would only be able to speculate as to the cause of death because the autopsy was limited in scope. He was confident that the death was unrelated to Mr. Voyten's employment in the coal mines because "the disease related to the coal mine exposure was so minimal that it would not ... have affected cardiac rhythm." He also cited the fact that "physiologic studies ... show[ed] that his lung function test and blood oxygen levels were normal at least as measured years before his death." (*Id.* at 33).

Dr. Tomashefski is Chairman of the Department of Pathology, MetroHealth Medical Center, and full Professor of Pathology at the Case Western Reserve University. He is Vice Chairman of the Department of Pathology at Case Western, is board certified in anatomic and clinical pathology, and is widely published in the field. (EX 3).

Dr. Gregory J. Fino

Dr. Fino reviewed the Miner's medical records and reported his findings in a report dated October 5, 2004. (EX 6). He concluded that Mr. Voyten had coal workers' pneumoconiosis. Dr. Fino disagreed that the Miner had shown hypoxemia in the arterial blood gas studies. Specifically, Dr. Fino stated that "all of the information in this case does not substantiate an opinion that this man had hypoxemia due to any type of lung disease that contributed to his death." He also opined that there was no evidence of clinical chronic obstructive pulmonary disease in the medical record. According to Dr. Fino,

> This man had numerous medical problems. He had rectal cancer metastatic to the lungs and bladder cancer. He had coronary artery disease and vascular disease that ultimately required abdominal aortic aneurysm surgery. There is no evidence, whatsoever, that lung disease, regardless of its cause, was a contributing or hastening factor to this man's death.

> ... The bottom line ... is that lung disease was never clinically significant. There was never any pulmonary impairment, and there was no evidence of hypoxemia.
>
> I can state with a reasonable degree of medical certainty that this man's simple coal workers' pneumoconiosis did not cause, contribute to or hasten this man's death. This man would have died as and when he did had he never stepped foot in the mines.

(EX 6). Dr. Fino also stated that the Miner did not suffer from pneumoconiosis. He nevertheless assumed that, even if Mr. Voyten suffered from the disease, there was no respiratory impairment. Dr. Fino is board certified in internal and pulmonary medicine and is a B-reader. (EX 7). His deposition was recorded on November 23, 2004, and he clarified his misstatement in the medical report that the Miner did not have pneumoconiosis, testifying that that was a mistake. Instead, Dr. Fino agreed that Mr. Voyten suffered from the disease. (EX 9 at 9).

Although he agreed that pneumoconiosis is a progressive disease, Dr. Fino opined that the Miner's pneumoconiosis did not progress. He also did not see clinical evidence of cor pulmonale in the medical record or by EKG. (*Id.* at 11). Nor could he discern from the records any pulmonary impairment. (*Id.* at 12–13, 21).

Although a limited autopsy was performed, Dr. Fino opined that the Miner's cancer was the cause of death. (*Id.* at 12–13). He opined that the Miner's pneumoconiosis played no role in death. (*Id.* at 23). He explained:

> [W]hat I can say with reasonable certainty is I don't find any evidence of a functional lung condition due to coal mine dust that in and of itself would have caused or contributed to his death.
>
> * * *
>
> [B]ased on the objective data that is available, there is no evidence that there was an impairment that would have caused disability in this man from a pulmonary standpoint.

(*Id.* at 25). On cross-examination, he further explained:

> [W]hen you look at the clinical information with respect to his lung function, there is nothing for me to suggest, regardless of what was seen pathologically, that lung disease would have hastened his death by that month or two months or four months.
>
> * * *
>
> But if we assume that it was his cancer, which I think based on the available information is the best bet [as the cause of death], there is no evidence that lung

disease had functionally impaired his overall system to have made him die sooner had it not been for his coal dust exposure.

Dr. Fino acknowledged on cross-examination that he never saw the autopsy slides and never treated Mr. Voyten. He also opined that the Miner's smoking likewise had no effect on his lung function, although he was questioned about complaints of shortness of breath or significant breathing problems in 1983. (*Id.* at 27). Dr. Fino was also questioned about the autopsy findings of cor pulmonale, in particular the dimensions of the right ventricle, and nevertheless asserted that he would not diagnose cor pulmonale, explaining that the testing and physical examinations showed no cor pulmonale clinically. (*Id.* at 32).

Entitlement

In order to establish entitlement to survivor's benefits in a claim filed on or after January 1, 1982, Claimant must establish that the Miner had pneumoconiosis arising out of coal mine employment and that the Miner's death was due to pneumoconiosis, that pneumoconiosis was a substantially contributing cause or factor leading to his death, that death was caused by complications of pneumoconiosis, or that the Miner had complicated pneumoconiosis. 20 C.F.R. §§ 718.1, 718.202, 718.203, 718.205(c), 718.304. *See Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993); *Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988).

There is no evidence of record that Mr. Voyten suffered from complicated pneumoconiosis. I must therefore evaluate the record as a whole to determine whether his death was due to pneumoconiosis, 20 C.F.R. § 718.205(c)(1), or whether pneumoconiosis was at least a "substantially contributing cause" of death. 20 C.F.R. § 718.205(c)(2); *Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1003, 13 B.L.R. 2-100 (3d Cir. 1989). Pneumoconiosis constitutes a "substantially contributing cause" of death if it serves to hasten death even briefly in any way. *Soubik v. Director*, *OWCP*, 366 F.3d 226, 232 n. 10, 23 B.L.R. 2-82 (3d Cir. 2004); *Mancia v. Director, OWCP*, 130 F.3d 579, 585, 21 B.L.R. 2-114 (3d Cir. 1997).

Upon review of the record evidence, as well as pertinent lay testimony, I find that Claimant has established by a preponderance of the evidence that the Miner's death was hastened by his pneumoconiosis. For the reasons set forth below, I thus find that Claimant has established entitlement to survivor's benefits by a preponderance of the record evidence.

Initially, I am mindful that Dr. Surinder S. Bajwa had treated Mr. Voyten for a number of years. The Secretary's regulations require an examination into the "nature of the relationship" between the physician and the patient, its duration and both frequency and extent of treatment. 20 C.F.R. § 718.104(d)(1)–(4).

20 C.F.R. § 718.104(d)(5). The Third Circuit has expressed a particular respect for the opinion of treating physicians. *See Balsavage v. Director*, *OWCP*, 295 F.3d 390, 396, 22 B.L.R. 2-386 (3d Cir. 2002). That court has also cautioned, however, that a physician's analysis must be based on adequate documentation. *See Lango v. Director*, *OWCP*, 104 F.3d 573, 576, 21 B.L.R. 2-12 (3d Cir. 1997). In like manner, the physician's conclusions should also be presented with an adequate rationale. In the final analysis, the credibility of the treating physician's opinion may

primarily rest on its "power to persuade." *Eastover Mining Co. v. Williams*, 338 F.3d 501, 513 (6th Cir. 2003). When provided the opportunity to review the Miner's medical records, Dr. Bajwa submitted a brief letter report that recited his conclusion that pneumoconiosis contributed to Mr. Voyten's death. This letter lacks an adequate analysis of the Miner's records and does not provide an explanation of Dr. Bajwa's conclusions.

Nevertheless, I find the opinions of Dr. Perper and Goldblatt to be persuasive with respect to the extent of the Miner's pneumoconiosis and the role of this disease in hastening the Miner's death. Moreover, while the Employer's experts opined that clinical testing, last done years before the Miner's death, did not demonstrate that he had suffered any pulmonary deficiency, and while there is a dearth of reliable clinical studies to document breathing problems during the final years of the Miner's life, I nevertheless credit the testimony of Mrs. Voyten and the Miner's son, Mr. John Voyten, who recounted that the Miner suffered some breathing difficulty. Lay testimony of this nature does not establish causation, but is highly relevant to the determination of the nature and extent of any pulmonary or respiratory insufficiency. *See Soubik*, 366 F.3d at 233–34. Given that the Miner suffered from pneumoconiosis, this testimony lends support to the Claimant's case. Moreover, the Miner used medications to address his breathing difficulties.

I also credit the diagnosis of cor pulmonale rendered by Drs. Goldblatt and Perper. Cor pulmonale, of course, is not evidence of cause of death. The significance of what was noted by Drs. Goldblatt and Perper, however, is that there was an adverse effect of the CWP that was diagnosed on the Miner's heart. The theory of this claim was that pneumoconiosis compromised the Miner's health so that he succumbed to his other conditions. This conclusion is an adequate rationale for the opinion that pneumoconiosis hastened the Miner's death, albeit to a slight degree. *See Zeigler Coal Co. v. Director*, *OWCP* [*Villain*], 312 F.3d 332, 334 (7th Cir. 2002). Indeed, Dr. Oesterling conceded at deposition that the Miner had a mildly compromised system. Although he strenuously opined that any pneumoconiosis was insignificant, Dr. Oesterling's conclusions do not persuasively eliminate the possibility that the Miner's pneumoconiosis hastened his death, albeit briefly. He did acknowledge that, in general, a compromised pulmonary system in general could hasten death.

Although Dr. Branscomb saw no documentation of any pulmonary insufficiency that would have hastened death, his conclusion is undermined to some extent by the testimony from the Claimant and the Miner's son that Mr. Voyten did suffer from breathing problems. Similarly, Dr. Fino was unable to discern any clinical documentation of pulmonary insufficiency. Again, however, the lay testimony and lifetime complaints of breathing difficulties provide substance to the opinions by Drs. Perper and Goldblatt.

It is well established that the fact-finder should not automatically accept the opinion of the autopsy prosector. Dr. Goldblatt's status thus does not, taken alone, entitle his conclusions to additional weight. Nevertheless, in this case Dr. Goldblatt's opinions are corroborated by the conclusions of Dr. Perper that were drawn from the latter's histological review. Dr. Goldblatt persuasively testified that his view of histological material could be enhanced by the fact that he was present at the point the material for the slides was taken. Moreover, I accept the opinions of Drs. Perper and Goldblatt with respect to the pathology evidence of the extent of the

pneumoconiosis that was observed. I credit Dr. Goldblatt's observation on gross examination of the extent of the nodules and macules that were seen during the gross examination.

I am also mindful of the impressive credentials of the Employer's experts, and have duly noted their qualifications. I have duly noted that Employer's experts are confident that the amount of pneumoconiosis is insignificant, that indicia of the disease could not be associated with the Miner's emphysema, and that the cancer from which Mr. Voyten suffered was not related to coal mine dust exposure. I have also accounted for Dr. Perper's statement on cross-examination that the Miner quit smoking fifty years before his death, which differs from the general consensus in the record that Mr. Voyten smoked for approximately thirty years at a rate of one-half to three-quarters of a pack of cigarettes per day, ending in the early 1980s. I have evaluated Dr. Perper's statement and find that it does not detract from his opinions as a reviewing pathologist about the nature and extent of the coal workers' pneumoconiosis present.

On this record, I find that the opinions of Claimant's experts establish that it is more likely than not that the Miner's coal workers' pneumoconiosis hastened his death. Given the findings on gross and histological examination, and in view of evidence that the Miner suffered from breathing problems, I consider the opinions of Drs. Goldblatt and Perper to be better reasoned and more persuasive than the conclusions of Drs. Oesterling and Tomashefski, who effectively deemed the Miner's pneumoconiosis insignificant, trivial or incidental.[3] *See generally Clark v. Karst-Robbins Corp.*, 12 B.L.R. 1-149 (1989)(*en banc*); *Lucostic v. United States Steel Corp.*, 8 B.L.R. 1-46 (1985). I find that Mrs. Voyten is entitled to survivor's benefits under the Act.

ATTORNEY FEE

No award of attorney's fees for services rendered to Claimant is made herein, as no application has been received. Thirty days are hereby allowed to Claimant's counsel for the submission of such an application. In its preparation, counsel's attention is directed to 20 C.F.R. §§ 725.365 and 366. A service sheet showing that service has been made upon all parties, including Claimant, must accompany the application. Parties have ten days following receipt of the application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

[3] I emphasize that the lay testimony does not trump the opinions from the Employer's pulmonologists. I have duly considered the conclusions of Drs. Branscomb and Fino about the lack of any clinical documentation of any pulmonary or respiratory difficulties. The more probative evidence in this case rests in the opinions of the pathologists for both parties who examined the slides first hand and could bring their expertise to bear. Of these, I credit the opinions from Drs. Goldblatt and Perper with respect to the nature and extent of the disease process they observed.

Finally, I have considered the death certificate, in which pneumoconiosis is implicated in the Miner's death. I have accorded it some, although not controlling, weight.

ORDER

The claim of ELEANORA M. VOYTEN for survivor's benefits under the Act is hereby GRANTED.

Accordingly, the CONSOLIDATION COAL COMPANY shall:

1. Pay ELEANORA M. VOYTEN benefits on her survivor's claim, with the onset of benefits commencing July 1, 2002.

2. Reimburse the Secretary of Labor for any payments made to Claimant, if any, and deduct such amounts from those ordered in Paragraph 1.

3. Pay Claimant or the Secretary of Labor, as appropriate, interest at the rate applicable under 20 C.F.R. § 725.608.

4. Pay Claimant's attorney, Robert L. Bilonick, Esquire, fees and expenses to be established in a Supplemental Decision and Order.

A

MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Avenue, NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).